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=> s "b72.3"
 658 "B72"
 2107088 "3"
L1 621 "B72.3"
 ("B72" (W) "3")

=> s tag(W)72
 8729 TAG
 3995 TAGS
 11532 TAG
 (TAG OR TAGS)
 114021 72
L2 395 TAG(W) 72

=> s tag72
L3 57 TAG72

=> s sialyl(W) TN
 2972 SIALYL
 5030 TN
 499 TNS
 5514 TN
 (TN OR TNS)
L4 204 SIALYL(W) TN

=> s 11 or 12 or 13 or 14
L5 1069 L1 OR L2 OR L3 OR L4

=> s immune(W) complex
 664365 IMMUNE
 32 IMMUNES
 664380 IMMUNE
 (IMMUNE OR IMMUNES)
 389095 COMPLEX
 112985 COMPLEXES
 460258 COMPLEX
 (COMPLEX OR COMPLEXES)
L6 19390 IMMUNE(W) COMPLEX

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=> s 17
444 "B72"
5371412 "3"
212 "B72.3"
("B72" (W) "3")
10529 TAG
3683 TAGS
12354 TAG
(TAG OR TAGS)
213500 72
263 TAG(W) 72
77 TAG72
2895 SIALYL
1 SIALYLS
2895 SIALYL
(SIALYL OR SIALYLS)
13217 TN
581 TNS
13784 TN
(TN OR TNS)
133 SIALYL(W) TN
132002 IMMUNE
6 IMMUNES
132004 IMMUNE
(IMMUNE OR IMMUNES)
991051 COMPLEX
575957 COMPLEXES

1226421 COMPLEX
(COMPLEX OR COMPLEXES)
8580 IMMUNE (W) COMPLEX
L8 4 L5 AND L6

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ENTER L# LIST OR (END):17-18

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PROCESSING COMPLETED FOR L8

L9 7 DUP REM L7-L8 (2 DUPLICATES REMOVED)

=> d 19 1-7 bib ab

L9 ANSWER 1 OF 7 CA COPYRIGHT 2002 ACS
AN 133:361751 CA
TI Study of B72.3 combining sites by molecular modeling
and site-directed mutagenesis
AU Xiang, Jim; Srivamadan, Maheswaran; Rajala, Raju; Jia, Zongchao
CS Departments of Microbiology, Oncology, Saskatoon Cancer Center, University
of Saskatchewan, Saskatoon, SK, S7N 0W0, Can.
SO Protein Engineering (2000), 13(5), 339-344
CODEN: PRENE9; ISSN: 0269-2139
PB Oxford University Press
DT Journal
LA English
AB A B72.3 Fab/sTn2 complex was modeled from the known
structure of B72.3 Fab and the dimeric Tn-serine
cluster (sTn2). In the complex model, the side chains of 15 heavy- and
light-chain complementarity-detg. region (CDR) residues and the main
chains of two light-chain CDR residues contact the sTn2 epitope. Among 15
CDR residues which contact sTn2 in the model, two heavy-chain residues
(Ser95 and Tyr97) and light-chain CDR residue (Tyr96) have been confirmed
in a previous study. To test the accuracy of the computational model,
further site-directed mutagenesis was performed by alanine scanning on the
remaining 12 residues that are predicted in the model to have side-chain
interactions with sTn2. Of these 12 mutants, eight that are all from the
heavy-chain (His32Ala, Ala33Leu, Tyr50Ala, Ser52Ala, Asn52Ala, Asp56Ala,
Lys58Ala and Tyr96Ala) had significantly reduced sTn2 affinities, and four
consisting of three light-chain mutations (Asn32Ala, Trp92Ala and
Thr94Ala) and one heavy-chain mutation (His35Ala) retained wild-type sTn2
affinity. On the whole, this evidence suggests that the complex model,
although not perfect, is correct in many of its features. In a more
general vein, these results lend credibility to the computational modeling
approach for the study of the mol. basis of antigen-antibody complexes.

RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 7 CA COPYRIGHT 2002 ACS
AN 131:309818 CA
TI Tolerization of B-cell response to tumor and inhibition of immune
complex-mediated disease progression
IN Barbera-Guillem, Emilio; Nelson, M. Bud
PA Biocrystal Ltd., USA

instant
invention

SO PCT Int. Appl., 56 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|----|---|------|----------|-----------------|----------|
| PI | WO 9955363 | A1 | 19991104 | WO 1999-US9025 | 19990426 |
| | W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| | RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | | |
| | AU 9936656 | A1 | 19991116 | AU 1999-36656 | 19990426 |
| | EP 1073459 | A1 | 20010207 | EP 1999-918836 | 19990426 |
| | R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI | | | | |
| | US 6245752 | B1 | 20010612 | US 1999-299289 | 19990426 |

PRAI US 1998-83155P P 19980427
WO 1999-US9025 W 19990426

AB The authors disclose that a B-cell response to tumor-derived sol. antigens can promote disease progression via an **immune complex**-mediated mechanism. As an example, the spread of metastatic melanoma was retarded in mice depleted of B-cells. Depletion of IgG-producing cells in a breast cancer model allowed for a redn. in extra-regional metastasis. In addn., immunization with mucin led to an enhanced anti-mucin antibody response to tumor and acceleration of tumor growth. Also, the authors disclose a compn. comprised of a non-immunogenic carrier mol. to which is linked carbohydrate chains representing repeated, antigenic carbohydrate determinants derived from shed antigens of interest.

RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
AN 1992:7201 BIOSIS
DN BA93:7201
TI PHARMACOKINETICS AND IMMUNE RESPONSE OF IODINE-131 CHIMERIC MOUSE-HUMAN
B72.3 HUMAN GAMMA-4 MONOCLONAL ANTIBODY IN HUMANS.
AU KHAZAEI M B; SALEH M N; LIU T P; MEREDITH R F; WHEELER R H; BAKER T S;
KING D; SECHER D; ALLEN L; ET AL
CS UNIV. ALABAMA BIRMINGHAM, COMPREHENSIVE CANCER CENTER, L.B. WALLACE TUMOR
INST. 262B, UAB STATION, BIRMINGHAM, ALA. 35294.
SO CANCER RES, (1991) 51 (20), 5461-5466.
CODEN: CNREA8. ISSN: 0008-5472.

FS BA; OLD
LA English

AB Chimeric **B72.3**, composed of the V-regions of murine **B72.3** and the constant regions of human immunoglobulin G4 heavy and .kappa. light chain, was administered as a ¹³¹I-labeled conjugate to 12 patients with metastatic colon cancer. Seven of these patients had an antibody response after initial infusion, and the immune response was primary directed to the murine V-region, although a small proportion of the antibody response was directed to topographical epitopes requiring the presence of both murine V-region and human CH-1 and .kappa. constant regions (neo-epitopes). The pharmacokinetics included a plasma disappearance curve best fit by a two-compartmental model with an .alpha. t_{1/2} of 18 .+-. 7 h and a .beta. t_{1/2} of 224 .+-. 66 h. A second infusion of the same dose of ¹³¹I-chimeric **B72.3** was administered to four of these patients 8 wk after the first infusion. Two patients who had a high antibody response to initial infusion had an anamnestic antibody response, and the infused ch-**B72.3**

rapidly disappeared from the circulation with associated **immune complexes** and free ^{131}I in the plasma. One patient with no initial antibody response had no antibody response and identical pharmacokinetics on second infusion. One patient with a modest transient antibody response to initial infusion had no antibody response on second infusion and a modest shortening of plasma circulation. Thus, the human immunoglobulin G4 isotype chimeric **B72.3** monoclonal antibody has a plasma half-life 6 to 8 times as long as murine **B72.3** and retains considerable immunogenicity in some patients which can adversely affect repetitive infusions.

L9 ANSWER 4 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1990:416462 BIOSIS
DN BA90:77263
TI IN-VIVO FATE OF MONOCLONAL ANTIBODY **B72.3** IN PATIENTS WITH COLORECTAL CANCER.
AU COLCHER D; MILENIC D E; FERRONI P; CARRASQUILLO J A; REYNOLDS J C; ROSELLI M; LARSON S M; SCHLOM J
CS BUILD. 10 ROOM 807, LABORATORY TUMOR IMMUNOL. BIOL., NATIONAL CANCER INST., 900 ROCKVILLE PIKE, BETHESDA, MD. 20892.
SO J NUCL MED, (1990) 31 (7), 1133-1146.
CODEN: JNMEAQ. ISSN: 0161-5505.
FS BA; OLD
LA English
AB Radiolabeled **B72.3** (anti-**TAG-72**) has been selectively localized metastatic lesions in 70%-80% of the cases. Serum samples from 27 colorectal carcinoma patients who received iodine-131-(^{131}I) **B72.3** by i.v. administration were analyzed. Circulating immunoreactive antibody followed a biphasic clearance pattern. High-performance liquid chromatography (HPLC) and SDS-polyacrylamide gel electrophoresis demonstrated that ^{131}I -**B72.3** retained its integrity in the patients' sera. HPLC analysis also demonstrated the presence of **immune complexes** in the sera of 12 patients; this correlated with elevated levels of circulating **TAG-72**. Several different HAMA response patterns were detected in the 25 patients' sera that were analyzed; some patients developed HAMA as early as 5-7 days post-MAb injection. Higher doses of administered MAb **B72.3** correlated with the development of HAMA ($p = 0.007$). The presence of elevated levels of **TAG-72** in the patients' pre-inoculum serum was shown to correlate with the detection of lesions by gamma scanning. Serum **TAG-72** may serve as a criteria for patient selection for immunodiagnostic or immunotherapeutic procedures using MAb **B72.3**.

L9 ANSWER 5 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2
AN 1989:450028 BIOSIS
DN BA88:98300
TI PHARMACOKINETIC EVALUATION OF TECHNETIUM-99 METALLOTHIONEIN-CONJUGATED MOUSE MONOCLONAL ANTIBODY **B72.3** IN RHESUS MONKEYS.
AU BURCHIEL S W; HADJIAN R A; HLADIK W B; DROZYNSKI C A; TOLMAN G L; HABER S B; GALLAGHER B M
CS UNIV. N.M. COLL. PHARM., IMMUNOPHARMACOL. RADIOPHARM. LAB., ALBUQUERQUE, N.M. 87131.
SO J NUCL MED, (1989) 30 (8), 1351-1357.
CODEN: JNMEAQ. ISSN: 0022-3123.
FS BA; OLD
LA English
AB These studies were conducted to determine the biodistribution and pharmacokinetics of [^{99}Tc]metallothionein-conjugated **B72.3** ([Tc]MT-**B72.3**) in Rhesus monkeys (*Macaca mulatta*) that were performed as part of the preclinical evaluation of [Tc]MT-**B72.3**. The **B72.3**-MT conjugate was studied at three doses of **B72.3** ranging

from 0.03 mg/kg to 1 mg/kg to determine whether a relationship existed between the dose of total antibody administered intravenously and the biodistribution and clearance of the radiolabeled protein. Results indicated that [Tc]MT-**B72.3** distributes rapidly to central body cavity organs and that there was no difference in the rate of blood elimination for the three doses of **B72.3** studied. The terminal phase of blood elimination was found to be 26.2 .+- .6.1 hr for the combined groups of monkeys. Approximately one-half of injected 99mTc activity was recovered in the urine within 24 hr. A second purpose of these studies was to evaluate the overall immunogenicity of the mouse monoclonal **B72.3** IgG1 antibody in Rhesus monkeys. These results demonstrated that a single i.v. exposure to mouse monoclonal **B72.3** at doses of 0.3 mg/kg or greater elicited antibody production to **B72.3** in Rhesus monkeys within 3 wk. Analysis of [Tc]MT-**B72.3** biodistribution and clearance in monkeys with circulating levels of antibodies to **B72.3** (immunized monkeys) revealed that the liver was the primary site of clearance of the presumed **immune complex** and that blood elimination was greatly accelerated.

L9 ANSWER 6 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1987:253158 BIOSIS
DN BA84:6130
TI QUANTITATIVE ANALYSES OF SELECTIVE RADIOLABELED MONOCLONAL ANTIBODY LOCALIZATION IN METASTATIC LESIONS OF COLORECTAL CANCER PATIENTS.
AU COLCHER D; ESTEBAN J M; CARRASQUILLO J A; SUGARBAKER P; REYNOLDS J C;
BRYANT G; LARSON S M; SCHLOM J
CS LAB. TUMOR IMMUNOLOGY AND BIOLOGY, BUILDING 10, ROOM 8B13, BETHESDA,
MARYLAND 20892.
SO CANCER RES, (1987) 47 (4), 1185-1189.
CODEN: CNREA8. ISSN: 0008-5472.
FS BA; OLD
LA English
AB We have previously demonstrated, using in vitro assays, a high degree of selective binding of monoclonal antibody (MAb) **B72.3** for carcinomas of the colon, ovary, and breast versus normal adult tissues using in vitro assays. We report here a demonstration of selective tumor localization in colorectal cancer patients of i.v. administered 131I-labeled MAb **B72.3** immunoglobulin G prior to surgery. Radiolocalization indices (RI) were obtained by direct analyses of biopsy materials (i.e., cpm of 131I-labeled MAb per g of tumor versus cpm per g of normal tissues). Using as a "positive" localization, RI of 3 times greater than normal tissue (i.e., RI > 3.0), tumor lesions in various sites from 17 of 20 patients scored positive. In eight of these patients, all tumor lesions demonstrated RIs of > 3, while in five patients RIs of some lesions were > 10 and as high as 30 to 46. Seventy % (99 of 142) of tumor lesions showed RIs of > 3, while only 12 of 210 histologically confirmed normal tissues examined showed RIs of > 3. These tissues, moreover, were either adjacent to tumor or draining tumor masses, or, as in the case of two patients, apparently due to high levels of circulating **immune complexes** that were deposited in the spleen. Positive gamma scans (confirmed at surgery) were observed in 14 of 27 patients. An isotype-identical control immunoglobulin G was coinjected and showed RIs considerably lower than that of **B72.3**.
3. No toxicity or adverse reaction was observed with either MAb. These studies are among the most comprehensive to date concerning the definition of the actual delivery of radiolabeled MAb to carcinoma lesions versus a wide range of adjacent and distal normal tissues and lead the way for other diagnostic and potential therapeutic applications of this antibody either alone, or in combinations with other monoclonal antibodies.

L9 ANSWER 7 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1987:169391 BIOSIS

DN BA83:87832
 TI QUANTITATIVE AND QUALITATIVE ASPECTS OF RADIOLOCALIZATION IN COLON CANCER
 PATIENTS OF INTRAVENOUSLY ADMINISTERED MAB **B72.3**.
 AU ESTEBAN J M; COLCHER D; SUGARBAKER P; CARRASQUILLO J A; BRYANT G; THOR A;
 REYNOLDS J C; LARSON S M; SCHLOM J
 CS LABORATORY OF TUMOR IMMUNOLOGY AND BIOLOGY, NATIONAL CANCER INSTITUTE,
 NATIONAL INSTITUTES OF HEALTH, BETHESDA, MD. 20892, USA.
 SO INT J CANCER, (1987) 39 (1), 50-59.
 CODEN: IJCNAW. ISSN: 0020-7136.
 FS BA; OLD
 LA English
 AB Monoclonal antibody (MAb) **B72.3** has been previously shown, by in vitro assays, to have a high degree of specificity for carcinomas of the colon, ovary and breast versus normal adult tissues. **B72.3** IgG was labelled with ¹³¹I and injected i.v. into 20 patients with known or suspected colorectal cancer. All patients subsequently underwent surgical exploration, with tumor and selected normal tissues removed for staging purposes. The selective localization of ¹³¹I-MAb **B72.3** IgG was demonstrated in biodistribution studies in which the % ID/g of each tumor was compared with that of the normal tissues, thus providing a relative RI for each lesion. Of the tumor lesions, 70% (99/142) had an RI of at least 3 (i.e., 3 times greater uptake per gram than normal tissues), and 31% of the tumor lesions had RIs of over 10. Only 12 of 210 (6%) histologically normal tissues had RIs of > 3; either these tissues were adjacent to or draining tumor masses or, as in the case of 2 patients, the high RI values were apparently due to deposition of immune complexes in the splenic tissues. Several parameters were studied to determine factors that might influence MAb localization. Whereas tumors of all histologic types localized the MAb, 31% of the well-differentiated mucinous carcinomas displayed tumor-to-normal ratios greater than 10, while less than 5% of the lesions of other tumor types demonstrated similar localization. The expression of the antigen (**TAG-72**) detected by MAb **B72.3** in these tumors, as studied by immunohistochemical techniques using tissue sections, did not always correlate with the outcome of the MAb distribution. No differences in MAb uptake were observed among the carcinoma lesions from numerous anatomic locations, demonstrating the ability of i.v. administered **B72.3** to reach all the tumor sites. Furthermore, autoradiographic studies of tumors showed good penetration of the MAb into the medial areas of the tumors, regardless of their size.

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NEWS 5 Feb 19 Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 6 Mar 08 Gene Names now available in BIOSIS
NEWS 7 Mar 22 TOXLIT no longer available
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NEWS 21 Jun 10 PCTFULL has been reloaded
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L1 6826 SIALO?

=> s sialyl?
L2 6762 SIALYL?

=> s NeuAc
L3 1062 NEUAC

=> s sialic(W)acid
12743 SIALIC
1075409 ACID
281730 ACIDS
1200820 ACID
(ACID OR ACIDS)

L4 12674 SIALIC(W)ACID

=> s acetylneuraminic(W)acid
2283 ACETYLNEURAMINIC
1075409 ACID
281730 ACIDS
1200820 ACID
(ACID OR ACIDS)

L5 2254 ACETYLNEURAMINIC(W)ACID

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L6 NOT FOUND

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of L-numbers, enter DISPLAY HISTORY at an arrow prompt (>).

=> s 11 or 12 or 13 or 14 or 15
L6 24616 L1 OR L2 OR L3 OR L4 OR L5

=> s immune(W)complex
664365 IMMUNE
32 IMMUNES
664380 IMMUNE
(IMMUNE OR IMMUNES)
389095 COMPLEX
112985 COMPLEXES
460258 COMPLEX

(COMPLEX OR COMPLEXES)

L7 19390 IMMUNE(W) COMPLEX

=> s cancer or tumor or carcinoma
 401580 CANCER
 40388 CANCERS
 411991 CANCER
 (CANCER OR CANCERS)
 561492 TUMOR
 181812 TUMORS
 632962 TUMOR
 (TUMOR OR TUMORS)
 246256 CARCINOMA
 46749 CARCINOMAS
 258 CARCINOMATA
 262387 CARCINOMA
 (CARCINOMA OR CARCINOMAS OR CARCINOMATA)

L8 999637 CANCER OR TUMOR OR CARCINOMA

=> s 16 and 17 and 18
L9 8 L6 AND L7 AND L8

=> file ca

| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
|----------------------|------------------|---------------|
| FULL ESTIMATED COST | 5.81 | 6.02 |

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FILE COVERS 1907 - 1 Aug 2002 VOL 137 ISS 6
FILE LAST UPDATED: 1 Aug 2002 (20020801/ED)

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=> s 19
 17119 SIALO?
 7186 SIALYL?
 1286 NEUAC
 17886 SIALIC
 3320905 ACID
 1288837 ACIDS
 3783208 ACID
 (ACID OR ACIDS)

16971 SIALIC(W)ACID
 4415 ACETYLNEURAMINIC
 3320905 ACID
 1288837 ACIDS
 3783208 ACID
 (ACID OR ACIDS)
 4367 ACETYLNEURAMINIC(W)ACID
 132002 IMMUNE
 6 IMMUNES
 132004 IMMUNE
 (IMMUNE OR IMMUNES)
 991051 COMPLEX
 575957 COMPLEXES
 1226421 COMPLEX
 (COMPLEX OR COMPLEXES)
 8580 IMMUNE(W) COMPLEX
 153488 CANCER
 21273 CANCERS
 159688 CANCER
 (CANCER OR CANCERS)
 247876 TUMOR
 103893 TUMORS
 282694 TUMOR
 (TUMOR OR TUMORS)
 87950 CARCINOMA
 20729 CARCINOMAS
 157 CARCINOMATA
 93906 CARCINOMA
 (CARCINOMA OR CARCINOMAS OR CARCINOMATA)

L10 14 L6 AND L7 AND L8

=> dup rem
ENTER L# LIST OR (END):19-110

COST IN U.S. DOLLARS

| SINCE | FILE | TOTAL |
|-------|------|---------|
| ENTRY | | SESSION |
| | | |
| 18.98 | | 25.00 |

FULL ESTIMATED COST

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PROCESSING COMPLETED FOR L9
PROCESSING COMPLETED FOR L10
L11 20 DUP REM L9-L10 (2 DUPLICATES REMOVED)

=> d 111 1-20 bib ab

L11 ANSWER 1 OF 20 CA COPYRIGHT 2002 ACS
AN 136:231238 CA
TI The diagnosis, prevention, amelioration and/or treatment of disturbed
immune function induced by disturbed lipid metabolism
IN Castro Cabezas, Manuel; Van Dijk, Hans
PA Universitair Medisch Centrum Utrecht, Neth.; Universiteit Utrecht
SO PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------------|-------|-------|-----------------|-------|
| ----- | ----- | ----- | ----- | ----- |

PI WO 2002022160 A2 20020321 WO 2001-NL672 20010912
 W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
 CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
 GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
 LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL,
 PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG,
 US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
 DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
 BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
 EP 1186299 A1 20020313 EP 2000-203156 20000912
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
 IE, SI, LT, LV, FI, RO

PRAI EP 2000-203156 A 20000912
 US 2000-253465P P 20001128

AB Complement is recognized as an important, humoral defense system involved in the innate (nonspecific) recognition and elimination of microbial invaders, other foreign particles or mols., and antigen-antibody complexes from the body. The present invention makes use of the surprising notion that the handling of lipids by the body, rather than its antimicrobial activity, is the primary and most ancient function of the complement system. Consequently, atherosclerosis as obsd. in disorders assocd. with disturbed lipid metab. must be ascribed to either genetic or acquired defects in ancient (activatory and/or regulatory) complement components. The surprising notion is of considerable consequence to the treatment of diseases of the immune system and/or an infectious, autoimmune, neoplastic and/or hematol. disease related to complement-mediated lipid metab. and/or an underlying and/or related disease since lipids and **immune complexes** share the same transport pathway in the human body. Other implications of the same invention, based on the notion that lipoproteins and lymphocytes share the lymph pathway to arrive in the blood circulation, are that the lipid metabolizing system may be employed to effectively manipulate the immune system. Based on this aspect of the invention, novel oral vaccination and oral immunomodulation strategies are introduced as well.

L11 ANSWER 2 OF 20 CA COPYRIGHT 2002 ACS

AN 133:295353 CA

TI Measurement of **immune complex**-mediated activation involving shed antigens

IN Barbera-Guillem, Emilio; Nelson, M. Bud

instant inventors

PA Biocrystal Ltd., USA

SO PCT Int. Appl., 72 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|--|--|----------|-----------------|----------|
| ----- | ----- | ----- | ----- | ----- |
| PI WO 2000059540 | A1 | 20001012 | WO 2000-US8932 | 20000404 |
| W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU,
CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL,
IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA,
MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI,
SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ,
BY, KG, KZ, MD, RU, TJ, TM | RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF,
CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | |

PRAI US 1999-127689P P 19990405

US 2000-541747 A 20000403

AB The authors disclose biol. responses inducible by activation of Fc.gamma.RI-expressing cells by **immune complexes**

contg. shed antigen. In one example, the proliferative response of **tumor** cells to anti-mucin **immune complexes** is demonstrated. In a second example, the matrix invasion by **tumor** cells is shown to be enhanced in the presence of stromal cells (granulocytes and macrophages) and anti-mucin **immune complexes**. In addn., the authors disclose the use of assays comprising shed antigen, anti-shed antigen IgG, and Fc. γ .RI-expressing cells for the detn. of activators and inhibitors of these **immune complex**-driven responses.

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 3 OF 20 CA COPYRIGHT 2002 ACS
AN 131:309818 CA
TI Tolerization of B-cell response to **tumor** and inhibition of **immune complex**-mediated disease progression
IN Barbera-Guillem, Emilio; Nelson, M. Bud *instant mucus*
PA Biocrystal Ltd., USA
SO PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|------|----------|-----------------|----------|
| PI WO 9955363 | A1 | 19991104 | WO 1999-US9025 | 19990426 |
| W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE,
KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW,
MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR,
TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,
ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG,
CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 9936656 | A1 | 19991116 | AU 1999-36656 | 19990426 |
| EP 1073459 | A1 | 20010207 | EP 1999-918836 | 19990426 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, FI | | | | |
| US 6245752 | B1 | 20010612 | US 1999-299289 | 19990426 |
| PRAI US 1998-83155P | P | 19980427 | | |
| WO 1999-US9025 | W | 19990426 | | |

AB The authors disclose that a B-cell response to **tumor**-derived sol. antigens can promote disease progression via an **immune complex**-mediated mechanism. As an example, the spread of metastatic melanoma was retarded in mice depleted of B-cells. Depletion of IgG-producing cells in a breast **cancer** model allowed for a redn. in extra-regional metastasis. In addn., immunization with mucin led to an enhanced anti-mucin antibody response to **tumor** and acceleration of **tumor** growth. Also, the authors disclose a compn. comprised of a non-immunogenic carrier mol. to which is linked carbohydrate chains representing repeated, antigenic carbohydrate determinants derived from shed antigens of interest.

RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 4 OF 20 CA COPYRIGHT 2002 ACS
AN 129:174688 CA
TI Stimulation of an immune response with antibodies labeled with the .alpha.-galactosyl epitope
IN Leung, Shui-On; Qu, Zhengxing
PA Immunomedics, Inc., USA
SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2

DT Patent
LA English
FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|---|--|----------|-----------------|----------|
| PI | WO 9834957 | A1 | 19980813 | WO 1998-US1976 | 19980206 |
| | W: | AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | |
| | RW: | GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG | | | |
| | AU 9860543 | A1 | 19980826 | AU 1998-60543 | 19980206 |
| | EP 1007569 | A1 | 20000614 | EP 1998-903896 | 19980206 |
| | R: | AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI | | | |
| | US 6090381 | A | 20000718 | US 1998-20299 | 19980206 |
| | JP 2001512438 | T2 | 20010821 | JP 1998-534822 | 19980206 |
| PRAI | US 1997-37908P | P | 19970211 | | |
| | WO 1998-US1976 | W | 19980206 | | |
| AB | The authors disclose a method for stimulation of humoral and cellular immune responses against tumor cells and infectious agents using an antibody that contains at least one .alpha.-galactosyl epitope. Such an antibody is capable of forming a complex with cells that express the target epitope and with xenoreactive antibodies that bind .alpha.-galactosyl epitopes. Suitable antibodies include mols. that contain at least one engineered glycosylation site in the const. region of the heavy chain. | | | | |

L11 ANSWER 5 OF 20 CA COPYRIGHT 2002 ACS

AN 129:310887 CA
TI **Tumor** necrosis factor formation inhibitors inhibiting selectins
IN Kawarai, Hiroko; Koike, Haruhiko; Tojo, Shinichiro
PA Sumitomo Pharmaceuticals Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 4 pp.

CODEN: JKXXAF

DT Patent
LA Japanese
FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|----|--|------|----------|-----------------|----------|
| PI | JP 10259140 | A2 | 19980929 | JP 1997-86122 | 19970318 |
| AB | The inhibitors, useful for treatment of diseases where TNF is involved, e.g. septicemia, rheumatoid arthritis, Kawasaki disease, ulcerative colitis, SLE, Behcet's disease, rejection in marrow transplantation, multiple organ failure, etc., contain anti-selectin antibodies or selectin-binding sugars. Anti-P-selectin monoclonal antibody ARP2-4 or sialyl-Lewis X deriv. [Et 5-acetamido-3,5-dideoxy-.alpha.-D-glycero-D-galacto-2-(nonulopyranosylonic acid)-(2-3)-O-(.beta.-D-galactopyranosyl)-(1-4)-O-[(.alpha.-L-fucopyranosyl-(1-3)-O]-(2-acetamido-2-deoxy-.beta.-D-glucopyranosyl)-(1-3)-O-.beta.-D-galactopyranoside sodium salt] significantly decreased TNF of BALF of rats with immune complex -induced pulmonary injury. | | | | |

L11 ANSWER 6 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

AN 2002:79922 BIOSIS
DN PREV200200079922
TI Method for increasing the sensitivity of assays for target ligand.
AU Linsley, P. S.; Ochs, V; Horn, D.; Brown, J. P.
CS Seattle, Wash. USA
ASSIGNEE: ONCOGEN LIMITED PARTNERSHIP

PI US 5646002 July 8, 1997
SO Official Gazette of the United States Patent and Trademark Office Patents,
(July 8, 1997) Vol. 1200, No. 2, pp. 1248.
ISSN: 0098-1133. *checked NR*
DT Patent
LA English

L11 ANSWER 7 OF 20 CA COPYRIGHT 2002 ACS
AN 125:162779 CA
TI Human **carcinoma** antigen (HCA), HCA antibodies, HCA immunoassays
and methods of imaging
IN Codington, John F.; Haavik, Svein
PA Epigen, Inc., USA
SO U.S., 34 pp., Cont.-in-part of U.S. Ser. No. i14,450, abandoned.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 3

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|----|------------|------|----------|-----------------|----------|
| PI | US 5545532 | A | 19960813 | US 1994-192840 | 19940207 |
| | US 5693763 | A | 19971202 | US 1995-468276 | 19950606 |
| | US 5808005 | A | 19980915 | US 1995-484061 | 19950606 |

PRAI US 1993-14450 19930205
US 1994-192840 19940207
AB A glycoprotein antigen which is generally characteristic of human carcinomas, regardless of the tissue assocd. with the carcinoma, is human **carcinoma** antigen (HCA), which is generally not present on normal human cells. Immunodeterminant-contg. fragments of HCA substantially sepd. from elements of HCA's naturally occurring environment are also disclosed. HCA is generally characterized by: (a) a mol. wt. in excess of 750,000; (b) carbohydrate moieties characteristic of mucin-type glycoproteins and comprising a relatively high proportion of **sialic acid**, galactose, and N-acetylgalactosamine residues (e.g., at least 50% of the carbohydrate residues are **sialic acid**, galactose, or N-acetylgalactosamine residues); (c) an isoelec. point below pH 3.0; (d) presence generally on human **carcinoma** cells; (e) absence generally from non-transformed human cells; (f) at least one immunodeterminant that specifically reacts with anti-murine epiglycanin antibody AE3; and (g) general insol. in aq. fluids (e.g., a phosphoric acid or an HCl soln.) below pH 2.0. Also disclosed are antibodies to HCA, immunoassays for HCA, and in vivo imaging using HCA-binding antibodies. *check*

L11 ANSWER 8 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE
1
AN 1996:270377 BIOSIS
DN PREV199698826506
TI Characterization of two glycolipid:alpha-2-3sialyltransferases, SAT-3 (CMP-**NeuAc**:nLcOse4Cer alpha-2-3sialyltransferase) and SAT-4 (CMP-**NeuAc**:GgOse4Cer alpha-2-3sialyltransferase), from human colon **carcinoma** (Colo 205) cell line.
AU Basu, Shib Sankar; Basu, Manju; Li, Zhixiong; Basu, Subhash (1)
CS (1) Dep. Chemistry Biochem., Univ. Notre Dame, Notre Dame, IN 46556 USA
SO Biochemistry, (1996) Vol. 35, No. 16, pp. 5166-5174.
ISSN: 0006-2960.
DT Article
LA English

AB **Sialyltransferase** activities, SAT-3 (CMP-**NeuAc**:nLcOse4Cer alpha-2-3sialyltransferase) and SAT-4 (CMP-**NeuAc**:GgOse4Cer alpha-2-3sialyltransferase), in Colo 205 cells catalyze the transfer of **sialic acid** to the terminal galactose of GlcNAc- and GalNAc-containing glycolipid substrates, respectively.

Competition kinetic studies with nLcOse4Cer and GM1 as substrates in a **sialyltransferase** assay show that these two activities are catalyzed by two different catalytic entities. The two enzymes were co-solubilized with taurocholate and resolved by DEAE-Cibacron Blue-Sepharose column chromatography into two elution peaks. The column eluent with SAT-3 activity failed to transfer **sialic acid** to asialo alpha-1-acid glycoprotein, indicating that this enzyme is different from the **sialyltransferase** (ST3N) that synthesizes **NeuAc**-alpha-2-3Gal linkage in asparagine-linked oligosaccharides of glycoprotein. However, SAT-3 activity can be immunoprecipitated with a polyclonal antibody produced against a protein expressed in Escherichia coli as GST-fusion protein from an ECB cDNA homolog of an alpha-2-3sialyltransferase (SAT-3 or STZ) that has been cloned from human melanoma cell and human placenta. Thus a concentration-dependent decrease in the residual SAT-3 activity relative to SAT-4 activity was observed in the supernatant after precipitation of the **immune complex**. Expression of SAT-3 (STZ) cDNA was also detected in Colo 205 cell by RTPCR, followed by sequence analysis of the RT-PCR product. Characterization of the catalytic reaction products of SAT-3 and SAT-4 with thin-layer chromatography, sialidase treatment, and binding to specific antibodies indicates that both SAT-3 and SAT-4 catalyze the formation of alpha-2-3 linkage between **sialic acid** and terminal galactose of glycolipid substrates.

L11 ANSWER 9 OF 20 CA COPYRIGHT 2002 ACS
AN 125:84117 CA
TI Characterization of a T cell line specific to an anti-Id antibody related to the carbohydrate antigen, **sialyl SSEA-1**, and the immunodominant T cell antigenic site of the antibody
AU Tsuyuoka, Kiyotaka; Yao, Kazuhiro; Hirashima, Kunimi; Ando, Shoji; Hanai, Nobuo; Saito, Hiromitsu; Yamasaki, Motoo; Takahashi, Katsutoshi; Fukuda, Yoshihiro; et al.
CS Research Institute, Aichi Cancer Center, Nagoya, Japan
SO Journal of Immunology (1996), 157(2), 661-669
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English
AB The stage-specific embryonic Ag-1 (SSEA-1) is a carbohydrate Ag and regarded as an onco-developmental Ag. **Sialyl SSEA-1** Ag, the **sialylated** form of SSEA-1, is frequently expressed in human **cancer** cells as well as in murine **cancer** cells. A mAb, FH-6, was shown to specifically recognize the Ag. We have generated five anti-Id Abs directed to the paratope-related idiotypes of the FH-6 Ab. One of these anti-Id Abs, Id-F2, increased the survival of host mice that were inoculated with Meth-A cells expressing the **sialyl SSEA-1** Ag. To clarify the exact mechanism underlying the antitumor effect of the anti-Id Ab, we established a T cell line that recognized Id-F2 in assocn. with MHC class II mols. The T cell line was CD4+V β 8+, and produced IL-2, exhibiting helper activity for B cells. The VH CDR2 region of the Id-F2 amino acid sequences turned out to be strongly immunogenic to T cells. When the **immune complexes**, consisting of the **sialyl SSEA-1** Ag, FH-6, and Id-F2, were formed at the Meth-A cell-surface, the T cell line showed a strong proliferative response. The possible roles played by such T cell subsets in the anti-**tumor** effect are discussed.

L11 ANSWER 10 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1996:383104 BIOSIS
DN PREV199699105460
TI Characterization of a T cell line specific to an anti-Id antibody related to the carbohydrate antigen, **Sialyl SSEA-1**, and the immunodominant T cell antigenic site of the antibody.

AU Tsuyuoka, Kiyotaka; Yago, Kazuhiro; Hirashima, Kunimi; Ando, Shoji; Hanai, Nobuo; Saito, Hiromitsu; Yamasaki, Motoo; Takahashi, Katsutoshi; Fukuda, Yoshihiro; Nakao, Kazuwa; Kannagi, Reiji (1)
CS (1) Lab. Experimental Pathol., Res. Inst., Aichi Cancer Cent., 1-1 Kanokoden, Chikusaku, Nagoya, 464 Japan
SO Journal of Immunology, (1996) Vol. 157, No. 2, pp. 660-669.
ISSN: 0022-1767.
DT Article
LA English
AB The stage-specific embryonic Ag-1 (SSEA-1) is a carbohydrate Ag and regarded as an onco-developmental Ag. ~~Sialyl~~ SSEA-1 Ag, the ~~sialylated~~ form of SSEA-1, is frequently expressed in human ~~cancer~~ cells as well as in murine ~~cancer~~ cells. A mAb, FH-6, was shown to specifically recognize the Ag. We have generated five anti-id Abs directed to the paratope-related idiotopes of the FH-6 Ab. One of these anti-id Abs, Id-F2, increased the survival of host mice that were inoculated with Meth-A cells expressing the ~~sialyl~~ SSEA-1 Ag. To clarify the exact mechanism underlying the antitumor effect of the anti-Id Ab, we established a T cell line that recognized Id-F2 in association with MHC class II molecules. The T cell line was CD4+V-beta-8+, and produced IL-2, exhibiting helper activity for B cells. The V-H CDR2 region of the Id-F2 amino acid sequences turned out to be strongly immunogenic to T cells. When the ~~immune complexes~~, consisting of the ~~sialyl~~ SSEA-1 Ag, FH-6, and Id-F2, were formed at the Meth-A cell-surface, the T cell line showed a strong proliferative response. The possible roles played by such T cell subsets in the anti-tumor effect are discussed.

L11 ANSWER 11 OF 20 CA COPYRIGHT 2002 ACS
AN 118:167305 CA
TI Requirements for leukocyte adhesion molecules in nephrotoxic nephritis
AU Mulligan, Michael S.; Johnson, Kent J.; Todd, Robert F., III; Issekutz, Thomas B.; Miyasaka, Masayuki; Tamatani, Takuya; Smith, C. Wayne; Anderson, Donald C.; Ward, Peter A.
CS Med. Sch., Univ. Michigan, Ann Arbor, MI, 48109, USA
SO J. Clin. Invest. (1993), 91(2), 577-87
CODEN: JCINAO; ISSN: 0021-9738
DT Journal
LA English
AB Requirements for leukocyte adhesion mols. as well as cytokines have been det. in the rat model of acute nephrotoxic nephritis. Proteinuria (at 24 h) and neutrophil accumulation in renal glomeruli (at 6 h) have been used as the endpoints. For full accumulation in glomeruli of neutrophils as well as full development of proteinuria, requirements have been demonstrated for TNF.alpha., (but not IL-1), CD11b (but not CD11a), very late arising-4 (CD49d/CD29), and intercellular adhesion mol.-1 but not endothelial leukocyte adhesion mol.-1 (E-selectin). By immunohistochem. approaches, infusion of antibody to glomerular basement membrane induced glomerular upregulation of intercellular adhesion mol.-1, endothelial leukocyte adhesion mol.-1, and vascular adhesion mol.-1. Treatment of rats with anti-TNF.alpha. or sol. recombinant human TNF receptor-1 blocked this expression. Renal arterial infusion of TNF.alpha. induced glomerular expression of all three endothelial adhesion mols., but infusion of IL-1.beta. did not. These data suggest that, in neutrophil and complement-dependent anti-glomerular basement membrane-induced acute nephritis in rats, there are selective requirements for cytokines, .beta.1 and .beta.2 integrins, and endothelial adhesion mols. These requirements contrast with those found in other vascular beds in which complement and neutrophil-induced vascular injury has been induced by deposition of ~~immune complexes~~.

L11 ANSWER 12 OF 20 CA COPYRIGHT 2002 ACS
AN 116:5088 CA

TI Recombinant gp120 specifically enhances **tumor** necrosis factor-.alpha. production and Ig secretion in B lymphocytes from HIV-infected individuals but not from seronegative donors
AU Rieckmann, Peter; Poli, Guido; Fox, Cecil H.; Kehrl, John H.; Fauci, Anthony S.
CS Lab. Immunoregul., Natl. Inst. Allergy Infect. Dis., Bethesda, MD, 20892, USA
SO J. Immunol. (1991), 147(9), 2922-7
CODEN: JOIMA3; ISSN: 0022-1767
DT Journal
LA English
AB The effect of recombinant protein from the envelope (gp120) of the HIV on B lymphocytes purified from either HIV-infected individuals or healthy seroneg. controls was examd. B cells from peripheral blood and lymph nodes of HIV-infected individuals spontaneously secreted TNF-.alpha.; this secretion was augmented by the presence of gp120, whereas B cells from healthy seroneg. donors failed to secrete/ significant levels of TNF-.alpha. in the presence or absence of gp120. In a coculture system of B cells and chronically HIV-infected T cells (ACH-2), where viral expression is largely mediated by TNF-.alpha., gp120 increased virus expression only if the B cells were obtained from HIV-infected individuals. The effects of gp120 on viral expression in this system were not mediated via CD4 receptor binding/or FcR binding of anti gp120-gp120 **immune complexes**. Besides its effect on cytokine prodn., gp120 also stimulated Ig secretion in B cells from HIV-infected individuals, but not from normal donors. Finally, it was demonstrated by in situ hybridization that germinal centers of lymph nodes from HIV-infected individuals contain large amts. of HIV RNA that is in close proximity to germinal center B cells. These findings suggest that the hyperplastic germinal centers of lymph nodes provide an unique environment for virus expression and accumulation where gp120 stimulates B cells to secrete HIV inductive cytokines, such as IL-6 and TNF-.alpha., and thereby further enhances virus expression in infected cells in a paracrine manner.

L11 ANSWER 13 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1992:98280 BIOSIS
DN BA93:54830
TI ACUTE PHASE REACTANTS AND CIRCULATING **IMMUNE COMPLEXES** IN PATIENTS WITH OVARIAN **CARCINOMA**.
AU DOBRYSZYCKA W; GERBER J; ZUWALA-JAGIELLO J; UJEC M
CS DEP. PHRM. BIOCHEM., MED. ACAD., SZEWSKA 38, 50-139 WROCLAW, POL.
SO ARCH IMMUNOL THER EXP, (1991) 39 (1-2), 41-50.
CODEN: AITEAT. ISSN: 0004-069X.
FS BA; OLD
LA English
AB Serum levels of haptoglobin (HP), **sialic acid** total (NAN) and lipid-bound (NAL), seromucoid (SER), its content in total protein (%SER), as well as circulating **immune complexes** (CIC), were measured in sera of women with ovarian **carcinoma**, prior to their treatment and through the course of chemotherapy, remission and recurrence of malignancy, respectively. Control groups consisted of healthy women and patients with benign **tumors** (ovarian cysts and uterine myomas). Pretreatment measurements of acute phase reactants discriminated **cancers** (FIGO stages I+II, III, IV) from healthy group, however differences between benign **tumors** and stages of ovarian **cancer** were not so distinct. Changes in the examined parameters (acute phase reactants) indicated satisfactorily a response to the administered chemotherapy and early signs of the progression of the disease. Because of great variations in serum CIC concentrations, they were found to be of no value either in diagnosis or in the surveillance of the disease status.

AN 116:120482 CA
 TI Effect of .beta.-carotene, canthaxanthin and vitamin A on Ehrlich ascites cell bearing mice
 AU Maity, Putul; Saha, Sandip; Chowdhury, T; N. Roy
 CS Dep. Cell Biol., Chittaranjan Natl. Cancer Inst., Calcutta, 700 026, India
 SO Indian J. Cancer Chemother. (1991), 13(1), 11-14
 CODEN: ICCHD2; ISSN: 0970-2563
 DT Journal
 LA English
 AB In an attempt to explore the chemopreventative aspects of carotenoid pigments on neoplastic growth the authors have studied the effect of vitamin A, .beta.-carotene, and canthaxanthin on Ehrlich ascites **tumor** growth. Four parameters such as total **tumor** cell count, mean survival time, serum level of **sialic acid** and serum level of **immune complex** were studied to assess either inhibitory or enhancing effect of the carotenoids on **tumor** growth. The preliminary results of these studies indicated that .beta.-carotene and vitamin A have a **tumor** inhibitory effect and .beta.-carotene is most prominent in this respect. Canthaxanthin which is not a pro vitamin A show **tumor** enhancing effect.

L11 ANSWER 15 OF 20 CA COPYRIGHT 2002 ACS
 AN 112:18592 CA
 TI Anti-ganglioside GD1a monoclonal antibody MZ, MZ-producing cells, and MZ-containing reagent for diagnosis of **cancer** and systemic lupus erythematosus
 IN Shimada, Shizuo; Iwata, Daiji; Sato, Wakao
 PA Mitsui Toatsu Chemicals, Inc., Japan
 SO Eur. Pat. Appl., 13 pp.
 CODEN: EPXXDW
 DT Patent
 LA English

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| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|--|------|----------|-----------------|----------|
| PI | EP 307186 | A2 | 19890315 | EP 1988-308273 | 19880907 |
| | EP 307186 | A3 | 19900314 | | |
| | EP 307186 | B1 | 19940622 | | |
| | R: DE, FR, GB | | | | |
| | JP 01067198 | A2 | 19890313 | JP 1987-221862 | 19870907 |
| | JP 07116238 | B4 | 19951213 | | |
| | CA 1314246 | A1 | 19930309 | CA 1988-576560 | 19880906 |
| | US 5192662 | A | 19930309 | US 1988-241291 | 19880907 |
| | JP 08187081 | A2 | 19960723 | JP 1995-167874 | 19950612 |
| | JP 2635946 | B2 | 19970730 | | |
| PRAI | JP 1987-221862 | | 19870907 | | |
| AB | Disclosed are a novel anti-ganglioside GD1a monoclonal antibody (MZ) which is capable of recognizing ganglioside GD1a but is practically incapable of recognizing glycolipids GalCer, LacCer, <u>GB3</u> , Gb4, GA1, GA2, GM1, GM2, GM3, GD1b, GT1b, GQ1b, Fuc-GM1, nLC4, and sialosyl nLC4; MZ-producing cells; an MZ-contg. reagent; and a method for the detection or quantification of GD1a using the reagent, e.g. to diagnose cancer , systematic lupus erythematosus (SLE), and diseases resulting from org. injury to the nervous system. Mice were immunized 4-15 times with formalin-treated Salmonella minnesota, and monoclonal antibodies were produced by std. techniques by fusion of their spleen cells with mouse myeloma NS-1 and screening of the clones. Serum from cancer or SLE patients was adsorbed on protein A-polystyrene, immune complexes were <u>dissocd.</u> , the supernatant was extd. with CHCl3:MeOH (2:1), and the <u>GD1a</u> -contg. CHCl3 layer was dried and dissolved in buffered saline contg. 1% bovine serum albumin. This soln. was placed in microtiter plate wells and incubated with polystyrene-immobilized MZ for 3 | | | | |

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h at 37.degree., biotinylated MZ for 1 h at room temp., and peroxidase-labeled avidin for 1 h at room temp. After treatment with substrate, the absorbance was read and compared to a std. curve for GD1a detn. Cancer and SLE patients had higher GD1a levels than healthy subjects.

- L11 ANSWER 16 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1987:86994 BIOSIS
DN BA83:45572
TI IN-VITRO STUDY OF SOME LOCAL AND-OR GENERAL PATHOGENIC FACTORS INVOLVED IN STIMULATION OF METASTASIS.
AU ANDRIAN T; CURIGUT A; LEIBOVICI I; MURGOCI R; CURCA L; CHIRNOAGA A; MOCIULSCHI A; MIHAI C; IURASCU C
CS INST. ONCOLOGIC, BUCUREST, ROM.
SO REV CHIR ONCOL RADIOL O R L OFTALMOL STOMATOL SER ONCOL, (1986) 25 (2), 107-122.
CODEN: ONCODU.
FS BA; OLD
LA Romanian
AB A study was carried out on the biochemical, metabolic, hematologic and immunologic changes that take place in the serum, organs and tumours of C57Bl/6 mice and/or C57B/10 mice, grafted with Lewis lung carcinoma or melanoma B10, in the course of stimulated metastasis. The study included the testing of certain techniques for stimulating metastasis, determining of the quantitative changes in total sialic acid in the sera of cancerous animals, in the dynamics of the metastasis process, comparative study of energy metabolism in the metastatic cells and the primary tumour cells, quantitative changes of the ulceic acids in some immunoreactive organs (thymus, spleen) and the liver, coagulation factors in the course of metastasis, analysis of galactose receptors in tumoural cells, suppressor T and helper T lymphocytes, detection of the circulating immune complexes in the evolution of certain metastasizing tumours. The following results were obtained: In C57Bl/6 or C57B/10 mice, bearing Lewis carcinoma or melanoma/B16, total serum sialic acid levels were significantly higher than, normal, at all intervals studied; in the course of stimulated metastasis significant quantitative changes occurred in nucleic acids in the thymus; the metastatic cells utilize more than the primary tumour cells the inefficient pathway for the synthesis of energy, i.e. aerobic glycolysis; the presence of the primary tumour induces decrease in the number of thrombocytes and increase in fibrinogen level; galactose receptors were detected, extracted and purified from primary tumour cells and from pulmonary metastasis cells; the proportion of suppressor T lymphocytes significantly increases with the evolution of metastasis; similarly, an increase was observed in the circulating immune complexes.

- L11 ANSWER 17 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE
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AN 1986:124700 BIOSIS
DN BA81:35116
TI GANGLIOSIDES IN SERUM IMMUNE COMPLEXES FROM TUMOR-BEARING PATIENTS.
AU HAKANSSON L; FREDMAN P; SVENNERHOLM L
CS DEP. ONCOL., UNIV. HOSP. LINKOPING, LINKOPING, SWEDEN.
SO J BIOCHEM (TOKYO), (1985) 98 (3), 843-850.
CODEN: JOBIAO. ISSN: 0021-924X.
FS BA; OLD
LA English
AB Immune complexes in the serum of tumor/
-bearing patients were absorbed from whole blood or plasma on a protein A-Sepharose column. The adsorbed material was eluted, precipitated and

analyzed for gangliosides. All precipitates obtained from eight patients at different treatment occasions contained gangliosides at concentrations varying from 0.1 to 12.2 nmol **sialic acid**/mg protein. The compositions of gangliosides were similar among the patients, regardless of the type of **cancer**, and quite different from that of normal serum. Most (75-85% of total **sialic acid**) belonged to the gangliotetraose series, of which 26-33% was GM1, 26-34% GD1a, 8-17% GD1b, and 5-13% GT1b. However, the dominant ganglioside in normal serum, GM3, was present in only trace amounts, which ruled out a nonspecific adsorption of serum ganglioside by protein A-Sepharose. Similar results were obtained for whole blood and plasma treatments, and these results suggest a specific interaction between gangliosides of the gangliotetraose series and serum immunoglobulins, either by the gangliosides acting as antigens and forming **immune complexes** or by their binding to already formed complexes.

L11 ANSWER 18 OF 20 CA COPYRIGHT 2002 ACS
AN 101:35301 CA
TI Monoclonal antibodies to native noncollagenous bone-specific proteins
AU Stenner, Debra D.; Romberg, Robert W.; Tracy, Russell P.; Katzmann, Jerry
A.; Riggs, B. Lawrence; Mann, Kenneth G.
CS Dep. Intern. Med., Mayo Clin. Found., Rochester, MN, 55905, USA
SO Proc. Natl. Acad. Sci. U. S. A. (1984), 81(9), 2868-72
CODEN: PNASA6; ISSN: 0027-8424
DT Journal
LA English
AB Hybridoma technol. was used for prepn. of murine monoclonal antibodies of high titer against bone .gamma.-carboxyglutamate-contg. protein (Gla protein) and osteonectin. A procedure of immunization and hybridization similar to that already described (Katzmann, J. A. et al., 1981 and Foster, W. B. et al., 1982) was used. However, in contrast to earlier studies, mice were immunized with an unfractionated protein mixt. that had been extd. from bone under nondenaturing conditions. The ext. was labeled with ^{125}I by the chloramine-T method. After fusion and initial hybrid growth, screening was accomplished by a solid-phase radioimmunoassay with total ^{125}I -labeled bovine bone protein ext. as the tracer. The identities of antibody-bound ^{125}I -labeled proteins were assessed by dissolv. of the solid-phase **immune complex** in SDS and subsequent electrophoresis and autoradiog. Clones producing specific antibody to a single protein were selected by limiting diln. The identity of the proteins against which the specific antibodies were produced was confirmed by immunopptn., electrophoresis, and autoradiog. From 2 fusions, 30 pos. hybrids to bone-Gla protein were identified; 7 of these were subcloned and 1 was expanded as an ascites **tumor**. One hybrid population was pos. for osteonectin, a Mr 15,000 peptide, and for bone-Gla protein. By limiting diln., the osteonectin clone was selected and subsequently expanded as an ascites **tumor**. Titrn. curves made by using the resp. ^{125}I -labeled purified proteins show the ascites **tumors** to be producing antibody of high titer ($I_{50} = 10^{-6}$) for anti-bone-Gla protein and ($I_{50} = 10^{-5}$) for antiosteonectin. Both of the antibovine antibodies are cross-reactive with the corresponding human protein. Immobilized specific antbone-Gla protein was used to isolate human bone-Gla protein from an EDTA ext. of human cortical bone. Thus, the title method offers the possibility of developing a complete library of monoclonal antibodies against these and other bone-specific proteins.

L11 ANSWER 19 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1981:162298 BIOSIS
DN BA71:32290
TI A MULTI PARAMETRIC APPROACH TO **TUMOR** MARKERS DETECTABLE IN SERUM IN PATIENTS WITH **CARCINOMA** OF THE OVARY OR UTERINE CERVIX.
AU SARJADI S; DAUNTER B; MACKAY E; MAGON H; KHOO S K
CS DEP. OBSTET. GYNAECOL., UNIV. QUEENSL., CLIN. SCI. BUILD., R. BRISBANE

SO HOSP., BRISBANE, Q 4029, AUST.
GYNECOL ONCOL, (1980) 10 (2), 113-124.
CODEN: GYNOA3. ISSN: 0090-8258.

FS BA; OLD
LA English

AB A comparison of several serum **tumor** markers lactate dehydrogenase (LDH), **sialyltransferase** (ST) carcinoembryonic antigen (CEA), .beta.2 microglobulin (.beta.2M), .gamma.-chain fetal Hb (HbF), **immune complexes** (ImCp) and spermine (Spm) was made in patients with **carcinoma** of the ovary or cervix uteri and healthy control subjects. The greatest positive results were obtained with the markers LDH (40%) and .beta.2M (46%) for patients with **carcinoma** of the cervix and ovary, respectively. However, based on false positive results, the most suitable single marker for patients with **carcinoma** of the cervix was Spm (30%). When a multiparametric approach was taken, a combination of 4 of the 7 markers resulted in an increase in the positive results, that is, the **cancer** patients were positive for 1 of the 4 markers. This was 76% for patients with **carcinoma** of the cervix using the markers LDH, ST, Spm and .beta.2M and 79% for patients with **carcinoma** of the ovary using the markers ST, CEA, HbF and .beta.2M. All markers in both groups of **cancer** patients were elevated above control levels with the exception of ST which was decreased in patients with **carcinoma** of the ovary. ST, CEA and HbF were associated by multilinear regression analysis in the ovarian **cancer** group. As the ST approached normal levels CEA and HbF became elevated. Similarly there was a linear relationship between .beta.2M and ImCp demonstrating positively at similar times. [These findings are discussed in relation to the use of **tumor** markers in **cancer** management.]

L11 ANSWER 20 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1978:259713 BIOSIS
DN BA66:72210
TI IMMUNE RESPONSE TO RAUSCHER VIRUS INDUCED LEUKEMIA IN DBA MICE PART 2 CORRELATION BETWEEN ANTIGENIC EXPRESSION AND PATHOGENESIS.
AU TOTH F D; VACZI L
CS INST. MICROBIOL., UNIV. MED. SCH., 4012 DEBRECEN, HUNG.
SO NEOPLASMA (BRATISL), (1978) 25 (3), 265-272.
CODEN: NEOLA4. ISSN: 0028-2685.

FS BA; OLD
LA English

AB Previous studies demonstrated that the Rauscher virus induces a biphasic erythroleukemia in DBA mice, and the regression of the disease is connected with the appearance of antibody-dependent cellular cytotoxicity. Attempts were made to reveal the mechanism leading to the lethal exacerbation of the leukemia. In the sera of leukemic mice soluble **tumor**-specific antigen could be demonstrated in the stages of early progression and regression but not in the stage of exacerbation. The antigen was present in form of **immune complexes** with free antibodies in excess. The emergence of a new population of leukemia cells was observed during the stage of regression. On the surface of these cells the antigen receptor sites were masked by **sialic acid** which resulted in the loss of immunosensitivity and immunogenicity.